

# WEST Search History

DATE: Wednesday, September 25, 2002

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR</i>			
L8	opson\$9 same atp same administ\$9	0	L8
L7	antigen same atp same administ\$9	7	L7
L6	antigen same atp same (rbc or ghost\$4)	1	L6
L5	antigen same atp	415	L5
L4	antigen same atp same opson\$9	0	L4
L3	L2 and atp	7	L3
L2	L1 and antigen	13	L2
L1	(silverstein)[in] or (loiike) [in] or (divirgilia)[in]	400	L1

END OF SEARCH HISTORY

(FILE 'HOME' ENTERED AT 20:03:16 ON 25 SEP 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002  
L1 1375 S SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU  
L2 304 S L1 AND IMMUN?  
L3 11 S L1 AND (HISTOCOMPATIBILITY OR MHC)  
L4 8 DUP REM L3 (3 DUPLICATES REMOVED)  
L5 0 S (ANTIGEN (1N) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND (  
L6 15 S ANTIGEN (P) ATP (P) PRESENT? (P) APC  
L7 5 DUP REM L6 (10 DUPLICATES REMOVED)  
L8 0 S OPSON? (P) GHOST? (P) ATP  
L9 123 S OPSON? (P) ATP  
L10 45 DUP REM L9 (78 DUPLICATES REMOVED)  
L11 5 S L10 AND ANTIGEN

=> s 19 (P) administ?  
L12 0 L9 (P) ADMINIST?

=> s 16 (P) administ?  
L13 1 L6 (P) ADMINIST?

=> dis l13 ibib abs

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2002:184925 CAPLUS  
DOCUMENT NUMBER: 136:231233  
TITLE: A method for inducing or suppressing immunity  
comprising phagocytic particles and ATP receptors  
INVENTOR(S): Silverstein, Samuel C.; Loike, John D.; Divirgilio,  
Francesco  
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New  
York, USA  
SOURCE: PCT Int. Appl., 74 pp.  
CODEN: P1XXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020042	A1	20020314	WO 2001-US28171	20010907
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CP, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an **antigen** to class I or class II MHC receptors to induce immunity against the **antigen** in a subject having a disease. This invention also provides methods of delivering an **antigen** to a class I or class II MHC receptor to suppress immunity against the **antigen** in a subject having a disease. The method comprises: (a) filling particles with the **antigen** and **ATP** resulting in Ag/ATP-filled particles; (b) coating the Ag/ATP-filled particles with a ligand for an **antigen-presenting** cell; (c) incubating the ligand-coated Ag/ATP-filled particles with ligand-binding **antigen-presenting** cells under conditions permitting the APCs to bind to the particles and APC phagolysosomes to ingest the ligand-coated Ag/ATP-filled particles to facilitate delivery of the ingested **antigen** to class I or class II MHC and expression on the surface of the APC; (d) incubating the Ag-APCs of step (c) with lymphocytes previously removed from the subject having the disease; and (e) **administering** the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the **antigen**. The **antigen-presenting** cells can also be **administered** to induce or suppress immunity against the **antigen**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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NEWS 4 Apr 09 ZDB will be removed from STN  
NEWS 5 Apr 19 US Patent Applications available in IFICDB, IFIPAT, and IFIUDB  
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NEWS 7 Apr 22 BIOSIS Gene Names now available in TOXCENTER  
NEWS 8 Apr 22 Federal Research in Progress (FEDRIP) now available  
NEWS 9 Jun 03 New e-mail delivery for search results now available  
NEWS 10 Jun 10 MEDLINE Reload  
NEWS 11 Jun 10 PCTFULL has been reloaded  
NEWS 12 Jul 02 FOREGE no longer contains STANDARDS file segment  
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saved answer sets no longer valid  
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NEWS 18 Aug 08 NTIS has been reloaded and enhanced  
NEWS 19 Aug 19 Aquatic Toxicity Information Retrieval (AQUIRE)  
now available on STN  
NEWS 20 Aug 19 IFIPAT, IFICDB, and IFIUDB have been reloaded  
NEWS 21 Aug 19 The MEDLINE file segment of TOXCENTER has been reloaded  
NEWS 22 Aug 26 Sequence searching in REGISTRY enhanced  
NEWS 23 Sep 03 JAPIO has been reloaded and enhanced  
NEWS 24 Sep 16 Experimental properties added to the REGISTRY file  
NEWS 25 Sep 16 Indexing added to some pre-1967 records in CA/CAPLUS  
NEWS 26 Sep 16 CA Section Thesaurus available in CAPLUS and CA

NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d,  
CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),  
AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002  
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=> file medline caplus embase biosis  
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 20:03:47 ON 25 SEP 2002

FILE 'CAPLUS' ENTERED AT 20:03:47 ON 25 SEP 2002  
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FILE 'BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002  
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=> s silverstein S?/au or loiike J?/au or divirgilio F?/au  
L1 1375 SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU

=> s l1 and immun?  
L2 304 L1 AND IMMUN?

=> s l1 and (histocompatibility or MHC)  
L3 11 L1 AND (HISTOCOMPATIBILITY OR MHC)

=> dup rem l3  
PROCESSING COMPLETED FOR L3  
L4 8 DUP REM L3 (3 DUPLICATES REMOVED)

=> dis l4 1-8 ibib abs

L4 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2002:184925 CAPLUS  
DOCUMENT NUMBER: 136:231233  
TITLE: A method for inducing or suppressing immunity  
comprising phagocytic particles and ATP receptors  
INVENTOR(S): Silverstein, Samuel C.; Loike, John  
D.; Divirgilio, Francesco  
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New  
York, USA  
SOURCE: PCT Int. Appl., 74 pp.  
CODEN: PIXXD2

DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
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WO 2002020042	A1	20020314	WO 2001-US28171	20010907
W:	AB, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			

PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an antigen to class I or class II MHC receptors to induce immunity against the antigen in a subject having a disease. This invention also provides methods of delivering an antigen to a class I or class II MHC receptor to suppress immunity against the antigen in a subject having a disease. The method comprises: (a) filling particles with the antigen and ATP resulting in Ag/ATP-filled particles; (b) coating the Ag/ATP-filled particles with a ligand for an antigen-presenting cell; (c) incubating the ligand-coated Ag/ATP-filled particles with ligand-binding antigen-presenting cells under conditions permitting the APCs to bind to the particles and APC phagolysosomes to ingest the ligand-coated Ag/ATP-filled particles to facilitate delivery of the ingested antigen to class I or class II MHC and expression on the surface of the APC; (d) incubating the Ag-APCs of step (c) with lymphocytes previously removed from the subject having the disease; and (e) administering the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the antigen. The antigen-presenting cells can also be administered to induce or suppress immunity against the antigen.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 8 MEDLINE DUPLICATE 1  
ACCESSION NUMBER: 91037972 MEDLINE  
DOCUMENT NUMBER: 91037972 PubMed ID: 2172452  
TITLE: Murine cytotoxic T lymphocytes specific for herpes simplex virus type 1 recognize the immediate early protein ICP4 but not ICP0.  
AUTHOR: Martin S; Zhu X X; Silverstein S J; Courtney R J; Yao F; Jenkins F J; Rouse B T  
CORPORATE SOURCE: Department of Microbiology and Nutrition Research, Upjohn Company, Kalamazoo, Michigan 49001.  
CONTRACT NUMBER: AI 14981 (NIAID)  
CA 42460 (NCI)  
GM 38125 (NIGMS)  
+  
SOURCE: JOURNAL OF GENERAL VIROLOGY, (1990 Oct) 71 ( Pt 10) 2391-9. Journal code: 0077340. ISSN: 0022-1317.  
PUB. COUNTRY: ENGLAND: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199012  
ENTRY DATE: Entered STN: 19910208  
Last Updated on STN: 19980206  
Entered Medline: 19901204

AB Vaccinia virus recombinants expressing the herpes simplex virus type 1 (HSV-1) genes encoding ICP0 or ICP4 were used to identify the precise target antigen(s) of murine anti-viral cytotoxic T lymphocytes (CTL) specific for the non-structural immediate early proteins. These studies revealed that HSV-1-specific CTL, restricted to class I major histocompatibility complex genes of the H-2k haplotype but not the H-2d or H-2b haplotypes, would lyse autologous cells expressing ICP4. HSV-1-specific CTL derived from various mice strains failed to lyse target cells expressing ICP0. Calculation of the frequencies of H-2k-restricted virus-specific CTL demonstrated that approximately a third of the total HSV-1-specific response was directed against ICP4. Immunization of mice with either recombinant vaccinia virus or transfected L cells expressing ICP4 induced HSV-1-specific lymphoproliferation and delayed hypersensitivity but CTLs were not induced. More importantly, such immunized animals were unable to resist or control a subsequent challenge with virulent HSV-1.

L4 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 1988:450619 BIOSIS  
DOCUMENT NUMBER: BR35:91499  
TITLE: PROCESSING AND PRESENTATION OF ANTIGENS.  
AUTHOR(S): PERNIS B; SILVERSTEIN S C; VOGEL H J  
CORPORATE SOURCE: COLL. PHYS. SURG., COLUMBIA UNIV., NEW YORK, N.Y.  
SOURCE: PERNIS, B., S. C. SILVERSTEIN AND H. J. VOGEL (ED.). PROCESSING AND PRESENTATION OF ANTIGENS. XIV+324P. ACADEMIC PRESS, INC.: SAN DIEGO, CALIFORNIA, USA; LONDON, ENGLAND, UK. ILLUS. (1988) 0 (0), XIV+342P. ISBN: 0-12-551855-2.  
DOCUMENT TYPE: Book  
FILE SEGMENT: BR; OLD  
LANGUAGE: English

AB Papers in this volume are the work of immunologists, biochemists, cell biologists, and virologists, and should be of interest to researchers in these disciplines. These papers on the early stages of the immune response are grouped under headings such as endosomes, lysosomes, and recycling, presentation in the context of class I major histocompatibility complex (MHC) molecules, and interactions of antigens with class II MHC molecules. The remaining parts deal with macrophages and dendritic cells as accessory cells, antigen presentation by B cells, and what T cells see. Illustrations and graphs supplement the text, and an index is provided.

L4 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 1988:547499 CAPLUS  
DOCUMENT NUMBER: 109:147499  
TITLE: Localization of immune receptor recognition sites on major histocompatibility molecules through the analysis of H-2Kb mutants  
AUTHOR(S): Zeff, Richard A.; Kumar, P. Ajit; Geliebter, Jan; Nathenson, Stanley A.

CORPORATE SOURCE: Dep. Cell Biol., Albert Einstein Coll. Med., Bronx,  
NY, 10461, USA

SOURCE: Process. Presentation Antigens (1988), 263-72.  
Editor(s): Pernis, Benvenuto; Silverstein, Samuel  
C.; Vogel, Henry J. Academic: San Diego, Calif.  
CODEN: 56HSAQ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review with 33 refs. Studies on H-2Kb mutants suggest that interaction  
of these mols. with the T cell receptor app. occurs at sites that are  
formed from amino acid residues at localized positions on the .alpha.1 and  
.alpha.2 domains of the class I polypeptide.

L4 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1988:547498 CAPLUS

DOCUMENT NUMBER: 109:147498

TITLE: Recycling of histocompatibility molecules  
and antigen presentation

AUTHOR(S): Pernis, Benvenuto

CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, NY,  
10032, USA

SOURCE: Process. Presentation Antigens (1988), 247-59.  
Editor(s): Pernis, Benvenuto; Silverstein, Samuel  
C.; Vogel, Henry J. Academic: San Diego, Calif.  
CODEN: 56HSAQ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review with 44 refs. of the importance of the intracellular formation of  
complexes between immunogenic peptides and class I and class II  
histocompatibility antigens in antigen presentation to cytotoxic  
and helper T-lymphocytes.

L4 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1988:547495 CAPLUS

DOCUMENT NUMBER: 109:147495

TITLE: The role of MHC and amphipathic structures  
in T cell recognition: features determining  
immunodominance

AUTHOR(S): Berzofsky, Jay A.; Cease, Kemp B.; Berkower, Ira J.;  
Margalit, Hanah; Cornette, Jim; Spouge, John; Spencer,  
Cecilia; Buckenmeyer, Gail; Streicher, Howard; et al.

CORPORATE SOURCE: Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD,  
20892, USA

SOURCE: Process. Presentation Antigens (1988), 125-31.  
Editor(s): Pernis, Benvenuto; Silverstein, Samuel  
C.; Vogel, Henry J. Academic: San Diego, Calif.  
CODEN: 56HSAQ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review with 30 refs. of sperm whale myoglobin recognition by murine T  
cells. The immunodominance of the glutamyl residue 109 of the myoglobin  
is discussed with regard to its presence in an .alpha.-helix with  
amphipathic properties; amphipathic structures may be a prerequisite for  
antigen recognition by T cells. Dependence of T cell recognition of  
myoglobin on I-A and I-E class II histocompatibility antigens is  
also considered.

L4 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1988:627970 CAPLUS

DOCUMENT NUMBER: 109:227970

TITLE: The epitopes of influenza nucleoprotein recognized by  
cytotoxic T lymphocytes can be defined with short  
synthetic peptides

AUTHOR(S): Townsend, A. R. M.; Rothbard, J.; Gotch, F. M.;  
Bastin, J.; Bahadur, G.; Wraith, D.; McMichael, A. J.

CORPORATE SOURCE: Nuffield Dep. Clin. Med., John Radcliffe Hosp.,  
Headington/Oxford, OX3 9DU, UK

SOURCE: Process. Presentation Antigens (1988), 81-5.  
Editor(s): Pernis, Benvenuto; Silverstein, Samuel  
C.; Vogel, Henry J. Academic: San Diego, Calif.  
CODEN: 56HSAQ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review and discussion with 10 refs. of synthetic peptides that contain  
epitopes from influenza virus nucleoprotein and evidence that cytotoxic  
T-lymphocyte recognition of synthetic peptides is class I  
antigen-restricted.

L4 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1988:547493 CAPLUS

DOCUMENT NUMBER: 109:147493

TITLE: Pathways of viral antigen presentation in T lymphocyte  
recognition

AUTHOR(S): Braciale, T. J.; Morrison, L. A.; Henkel, T. J.;  
Braciale, V. L.

CORPORATE SOURCE: Sch. Med., Washington Univ., St. Louis, MO, 63110, USA

SOURCE: Process. Presentation Antigens (1988), 69-79.  
Editor(s): Pernis, Benvenuto; Silverstein, Samuel  
C.; Vogel, Henry J. Academic: San Diego, Calif.  
CODEN: 56HSAQ

DOCUMENT TYPE: Conference; General Review

LANGUAGE: English

AB A review with 22 refs. discussing evidence suggesting that class I and  
class II major histocompatibility complex-restricted  
T-lymphocytes recognize influenza antigens presented through distinctly  
different presentation pathways.

=> s (antigen (1N) present?) and (histocompatibility or MHC) and (red (1N) blood (1N) cell (1N) ghost)  
3 FILES SEARCHED...

L5 0 (ANTIGEN (1N) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND  
(RED (1N) BLOOD (1N) CELL (1N) GHOST)

=> s antigen (P) ATP (P) present? (P) APC

L6 15 ANTIGEN (P) ATP (P) PRESENT? (P) APC

=> dup rem l6  
PROCESSING COMPLETED FOR L6

L7 5 DUP REM L6 (10 DUPLICATES REMOVED)

=> dis l7 1-5 ibib abs

L7 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:184925 CAPLUS  
 DOCUMENT NUMBER: 136:231233  
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 INVENTOR(S): Silverstein, Samuel C.; Loike, John D.; Divirgilio,  
 Francesco  
 PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New  
 York, USA  
 SOURCE: PCT Int. Appl., 74 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

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PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an **antigen** to class  
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 suppress immunity against the **antigen** in a subject having a  
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**antigen** and ATP resulting in Ag/ATP-filled  
 particles; (b) coating the Ag/ATP-filled particles with a ligand  
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 APCs to bind to the particles and APC phagolysosomes to  
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 delivery of the ingested **antigen** to class I or class II MHC and  
 expression on the surface of the APC; (d) incubating the Ag-  
 APCs of step (c) with lymphocytes previously removed from the  
 subject having the disease; and (e) administering the incubated  
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REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS  
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 5 MEDLINE DUPLICATE 1  
 ACCESSION NUMBER: 97260241 MEDLINE  
 DOCUMENT NUMBER: 97260241 PubMed ID: 9106336  
 TITLE: Identification of two types of autoreactive T lymphocyte  
 clones cultured from cardiac allograft-infiltrating cells  
 incubated with recombinant mycobacterial heat shock protein  
 71.  
 AUTHOR: Liu K; Moliterno R A; Fu X F; Duquesnoy R J  
 CORPORATE SOURCE: Division of Transplantation Pathology, University of  
 Pittsburgh Medical Center, PA 15261, USA.  
 CONTRACT NUMBER: AI-23567 (NIAID)  
 SOURCE: TRANSPLANT IMMUNOLOGY, (1997 Mar) 5 (1) 57-65.  
 Journal code: 9309923. ISSN: 0966-3274.  
 PUB. COUNTRY: ENGLAND: United Kingdom  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199706  
 ENTRY DATE: Entered STN: 19970620  
 Last Updated on STN: 19990129  
 Entered Medline: 19970612

AB Recent studies in several laboratories have advanced the concept that  
 during cellular rejection, the allograft undergoes a stress response which  
 regulates the expression of stress proteins (or heat shock proteins, hsp)  
 and triggers the recruitment and activation of hsp-reactive lymphocytes.  
 In a rat model of heterotopic heart transplants we have found that  
 allograft-infiltrating lymphocytes respond to recombinant mycobacterial  
 hsp and irradiated syngeneic spleen cells as a source of self-APC  
 (**antigen-presenting** cells). This report describes T  
 cell clones generated by culturing ACI into Lewis rat cardiac  
 allograft-derived lymphocytes with mycobacterial hsp71, syngeneic spleen  
 cells and IL-2 (interleukin-2). Two groups of self-APC-reactive  
 T cell clones have been distinguished, all of them are CD3+, CD4+, CD8-.  
 One group is referred to as hsp71-dependent, autoreactive T cells because  
 these clones respond to self-APC but only in the presence of  
 hsp71. No reactivity is seen with mycobacterial hsp65 or when hsp71 is  
 tested with allo-PC from ACI donors or third-party APC from  
 Brown Norway (BN) rats. Treatment of hsp71 with trypsin, polymyxin B or  
 ATP-agarose chromatography abrogates the hsp71 effect thus  
 indicating that structurally intact hsp71 must interact with self-  
 APC which then activate hsp71-dependent, autoreactive T cells. The  
 second group of clones reacts to self-APC and while their  
 response does not require the presence of hsp71, their proliferation is  
 often augmented by hsp71 but not by hsp65. These hsp71-independent,  
 autoreactive clones do not respond to allo-APC from ACI donors  
 or third-party APC from BN rats. Polymyxin or trypsin treatment  
 had no significant effect on their proliferative responses. The data with  
 the anti-TCR-alpha beta monoclonal antibody R73 offer additional evidence  
 for two functionally different types of self-APC reactive CD4  
 cells infiltrating the allograft. R73 inhibits the proliferation of self-  
 APC induced responses of hsp71-independent clones as well as the  
 allo-APC induced responses of alloreactive T cell clones. In  
 contrast, this antibody augments the responses of hsp71-dependent T cells.  
 Moreover, these clones can also proliferate in response to self-  
 APC when hsp71 is substituted by R73. The hsp71-dependency of  
 self-APC reactive T cell reactivity represents a previously  
 unrecognized mechanism of cellular immunity to allografts. This mechanism  
 might be related to the peptide binding properties of hsp71 and the  
 ability of stress proteins to function as molecular chaperones in  
**antigen** processing.

L7 ANSWER 3 OF 5 MEDLINE DUPLICATE 2  
 ACCESSION NUMBER: 95276250 MEDLINE  
 DOCUMENT NUMBER: 95276250 PubMed ID: 7538819  
 TITLE: Role of extracellular adenosine triphosphate in the cytotoxic T-lymphocyte-mediated lysis of antigen presenting cells.  
 AUTHOR: Blanchard D K; Wei S; Duan C; Pericle F; Diaz J I; Djeu J Y  
 CORPORATE SOURCE: Department of Medical Microbiology and Immunology, University of South Florida College of Medicine, Tampa, USA.  
 CONTRACT NUMBER: AI-33674 (NIAID)  
 SOURCE: BLOOD, (1995 Jun 1) 85 (11) 3173-82.  
 Journal code: 7603509. ISSN: 0006-4971.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
 ENTRY MONTH: 199506  
 ENTRY DATE: Entered STN: 19950707  
 Last Updated on STN: 19970203  
 Entered Medline: 19950623

AB The lysis of antigen presenting cells (APCs) by cytotoxic T lymphocytes (CTLs) may be one mechanism whereby an immune response is downregulated by Staphylococcus superantigens. Disappearance of monocytes/macrophages from staphylococcal enterotoxin A (SEA)-activated peripheral blood mononuclear cell (PBMC) cultures, but not from control PBMC cultures was seen by flow cytometry. Recently, adenosine triphosphate (ATP) has been described as an effector molecule in CTL-mediated lysis of some murine tumor target cells. We have also shown that ATP caused the lysis of human macrophages, and that treatment of cells with interferon gamma (IFN gamma) rendered macrophages significantly more sensitive to ATP than untreated cells. To show that this purine nucleotide may play a role in modulating the immune system, we generated human CTLs that were stimulated with SEA, and used them as effector cells against SEA-pulsed autologous macrophages. CTLs were found to specifically lyse SEA-pulsed macrophages, while control, unpulsed, macrophages were unaffected. The addition of hexokinase, an enzyme that hydrolyzes ATP, significantly abrogated the killing of SEA-pulsed cells during the assay. In examining the mechanism of cytotoxicity, electron microscopy showed that macrophages incubated with both ATP and CTLs underwent necrosis, rather than apoptosis. From these results, it is suggested that ATP is released from CTLs during antigen presentation, and that IFN gamma-activated macrophages, which are inherently more sensitive to this mediator, are readily lysed and therefore removed from circulation, thus downregulating an immune response.

L7 ANSWER 4 OF 5 MEDLINE DUPLICATE 3  
 ACCESSION NUMBER: 95394018 MEDLINE  
 DOCUMENT NUMBER: 95394018 PubMed ID: 7664779  
 TITLE: Establishment of a cell line with features of early dendritic cell precursors from fetal mouse skin.  
 AUTHOR: Girolomoni G; Lutz M B; Pastore S; Assmann C U; Cavani A; Ricciardi-Castagnoli P  
 CORPORATE SOURCE: Laboratory of Immunology, Istituto Dermopatico dell'Immacolata, IRCCS, Rome, Italy.  
 SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1995 Aug) 25 (8) 2163-9.  
 Journal code: 1273201. ISSN: 0014-2980.  
 PUB. COUNTRY: GERMANY: Germany, Federal Republic of  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 199510  
 ENTRY DATE: Entered STN: 19951020  
 Last Updated on STN: 19951020  
 Entered Medline: 19951010

AB During ontogeny, the skin is progressively populated by major histocompatibility complex class II-negative dendritic cell (DC) precursors that then mature into efficient antigen-presenting cells (APC). To characterize these DC progenitors better, we generated myeloid cell lines from fetal mouse skin by infecting cell suspensions with a retroviral vector carrying an envAKR-mycMH2 fusion gene. These cells, represented by the line FSDC, displayed a dendritic morphology and their proliferation in serum-free medium was promoted by granulocyte/macrophage colony-stimulating factor (GM-CSF), but not macrophage-CSF. FSDC expressed strong surface-membrane ATP/ADPase activity, intracellular staining for 2A1 antigen, and a surface phenotype consistent with a myeloid precursor: H-2d,b+, I-Ad,b+, CD54+, CD11b+, CD11c+, 2.4G2+, F4/80+, CD44+, 2F8+, ER-MP 12-, Sca-1+, Sca-2+, NLDC-145-, B7.2+, B7.1-, J11d-, B220-, Thy-1-, and CD3-. FSDC stimulated poorly allogeneic or syngeneic T cells in the primary mixed-leukocyte reaction, and markedly increased this function after treatment with GM-CSF, GM-CSF and interleukin (IL)-4 or interferon-gamma (IFN-gamma); in contrast, stem cell factor, IL-1 alpha and tumor necrosis factor-alpha had no effect. Preculture with IFN-gamma was required for presentation of haptens to primed T cells in vitro. However, FSDC, even after cytokine activation, were less potent APC than adult epidermal Langerhans cells in both of the above assays. Finally, FSDC derivatized with haptens and injected either intravenously or subcutaneously could efficiently induce contact sensitivity responses in naive syngeneic mice. The results indicate that fetal mouse skin is colonized by myeloid precursors possessing a macrophage/immature DC-like surface phenotype and priming capacity in vivo. These cells need further differentiation and activation signals (e.g. cytokines) to express their antigen presenting potential in vitro.

L7 ANSWER 5 OF 5 MEDLINE DUPLICATE 4  
 ACCESSION NUMBER: 90063450 MEDLINE  
 DOCUMENT NUMBER: 90063450 PubMed ID: 2584924  
 TITLE: A peptide binding protein having a role in antigen presentation is a member of the HSP70 heat shock family.  
 AUTHOR: Vanbuskirk A; Crump B L; Margoliash E; Pierce S K  
 CORPORATE SOURCE: Department of Biochemistry, Molecular Biology and Cell Biology, Northwestern University, Evanston, Illinois 60208.  
 CONTRACT NUMBER: AI-12001 (NIAID)  
 SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1989 Dec 1) 170 (6) 1799-809.  
 Journal code: 2985109R. ISSN: 0022-1007.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199001  
ENTRY DATE: Entered STN: 19900328  
Last Updated on STN: 19970203  
Entered Medline: 19900108

AB The T cell recognition of globular protein **antigens** requires the processing and **presentation** of the antigen by Ia-expressing APCs. Processing is believed to involve the uptake of **antigen** into an acidic compartment where proteolysis occurs. The resulting peptides containing the T cell antigenic determinant are associated with Ia and **presented** at the cell surface to the specific T cells. The mechanisms by which antigenic peptides become associated with Ia is not known. We previously described a peptide binding protein of 72/74 x 10(3) Mr (PBP72/74) that plays a role in **antigen presentation** as shown by the ability of an antiserum raised in rabbits to affinity-purified PBP72/74 to block **presentation** of cytochrome c to a cytochrome c-specific T cell hybrid. Here we show that PBP72/74 is recognized by mAbs specific for members of the HSP70 family of proteins. In Western blots PBP72/74 is bound by mAb 7.10, specific for an evolutionarily conserved epitope of HSP proteins and by mAb N27, specific for both the constitutively expressed and inducible 72/73 x 10(3) Mr HSP70 proteins. In addition, PBP72/74 shares a second common feature of the HSP proteins, that of binding to **ATP**. Indeed, **ATP** causes the release of PBP72/74 from binding to a peptide fragment of cytochrome c (Pc 81-104) and PBP72/74 can be eluted from **ATP** columns by Pc 81-104. Finally, a portion of PBP72/74 is shown to be **present** on B cell surfaces by immunofluorescence staining. Thus, it appears that characteristics of the heat shock proteins are shared by a protein playing a role in **antigen presentation**, suggesting some commonality in function.

=> s opson? (P) ghost? (P) ATP  
L8 0 OPSON? (P) GHOST? (P) ATP

=> s opson? (P) ATP  
L9 123 OPSON? (P) ATP

=> dup rem 19  
PROCESSING COMPLETED FOR L9  
L10 45 DUP REM L9 (78 DUPLICATES REMOVED)

=> s l10 and antigen  
L11 5 L10 AND ANTIGEN

=> dis l11 1-5 ibib abs

L11 ANSWER 1 OF 5 MEDLINE  
ACCESSION NUMBER: 95359458 MEDLINE  
DOCUMENT NUMBER: 95359458 PubMed ID: 7632965  
TITLE: Influence of glutamine on the phenotype and function of human monocytes.  
AUTHOR: Spittler A; Winkler S; Gotzinger P; Oehler R; Willheim M; Tempfer C; Weigel G; Fugger R; Boltz-Nitulescu G; Roth E  
CORPORATE SOURCE: Department of Surgery, University of Vienna, Austria.  
SOURCE: BLOOD, (1995 Aug 15) 86 (4) 1564-9.  
Journal code: 7603509. ISSN: 0006-4971.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 199509  
ENTRY DATE: Entered STN: 19950921  
Last Updated on STN: 19980206  
Entered Medline: 19950914

AB Reduced concentrations of glutamine (GLN) in plasma and skeletal muscle, defective host defense systems, and a diminished expression of the HLA-DR **antigen** on monocytes are important diagnostic parameters for late post-injury sepsis. In this in vitro study, we investigated whether blood monocyte-derived macrophage **antigen** expression and function from healthy donors is influenced by GLN. Lowering the GLN concentration in culture medium from 2 mmol/L to 200 mmol/L reduced the expression of HLA-DR by 40% (P < .001) on monocyte-derived macrophages, and decreased tetanus toxoid-induced **antigen** presentation. In addition, low GLN levels downregulated the expression of intercellular adhesion molecule-1 (ICAM-1/CD54), Fc receptor for IgG (Fc gamma RI/CD64), and complement receptors type 3 (CR3; CD11b/CD18) and type 4 (CR4; CD11c/CD18). A correlation was found between the phagocytosis of IgG-sensitized ox erythrocytes or **opsonized** Escherichia coli and the decreased expression of Fc gamma RI and CR3. Monocyte expression of CD14, CD71, and Fc gamma RII/CD16 and capacity to phagocytose latex beads were not affected by altering the level of GLN. Depletion of GLN was associated with a significant reduction in cellular adenosine triphosphate (**ATP**), which may have influenced cell surface marker expression and phagocytosis. It remains to be seen whether these in vitro findings are of clinical significance in the treatment of sepsis.

L11 ANSWER 2 OF 5 MEDLINE  
ACCESSION NUMBER: 90234863 MEDLINE  
DOCUMENT NUMBER: 90234863 PubMed ID: 2184903  
TITLE: Regulation of autoimmune anti-platelet antibody-mediated adhesion of monocytes to platelet GPIIb/GPIIIa: effect of armed monocytes and the Mac-1 receptor.  
AUTHOR: Hymes K B; Schuck M P; Karparkin S  
CORPORATE SOURCE: New York University Medical School, NY 10016.  
CONTRACT NUMBER: HL-13336-17 (NHLBI)  
SOURCE: HL01821 (NHLBI)  
BLOOD, (1990 May 1) 75 (9) 1813-9.  
Journal code: 7603509. ISSN: 0006-4971.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 199006  
ENTRY DATE: Entered STN: 19900706  
Last Updated on STN: 19900706  
Entered Medline: 19900606

AB Platelet autoantigen-autoantibody-monocyte interaction was studied by utilization of a specific monoclonal antibody (MoAb) 10E5 to trap and immobilize the GPIIb-GPIIIa complex on microtiter plates. Peripheral blood mononuclear cells (PBMC) or purified monocytes formed distinct morphologic



clusters after incubation with immobilized antigen for 18 hours at 37 degrees C. PBMC of 18 and 19 patients with autoimmune thrombocytopenic purpura (ATP) formed  $48 \pm 6.8$  (SEM) clusters/well compared with  $7.4 \pm 1.0$  for control subjects,  $P$  less than .001. The number of clusters per well correlated inversely and exponentially with platelet count,  $r = -.8$ ,  $n = 21$ , indicating that the GPIIb-GPIIIa autoantigen is pathophysiologically relevant. Binding of ATP PBMC to immobilized GPIIb-GPIIIa could be inhibited by  $F(ab')_2$  fragments of immunoglobulin (Ig) G of ATP patients, indicating that monocyte IgG bound to autoantigen by its  $F(ab')_2$  domain. Optimal cluster formation could be obtained with normal monocytes if preincubated with ATP IgG but not with  $F(ab')_2$  fragments of ATP IgG, indicating that ATP IgG binds to monocytes by its  $F_c$  domain. Armed monocytes (ie, normal monocytes preincubated with ATP IgG) bound to immobilized autoantigen 5.8-fold greater than normal monocytes incubated with immobilized autoantigen opsonized with ATP IgG. Armed monocyte adhesion could be inhibited 81% from  $18.9 \pm 1.6$  to  $3.6 \pm 0.5$  clusters/well by prior fixation with 0.1% formalin, whereas fixation of IgG before arming of monocytes was not inhibitory. MoAb MM41, directed against the alpha m-chain of the Mac-1 adhesive protein receptor of monocytes, inhibited cluster formation by 79%. Thus, (1) armed monocyte interaction with autoantigen is considerably more effective than monocyte interaction with opsonized autoantigen; (2) armed monocyte interaction requires specific  $F(ab')_2$ -antigen recognition; and (3) monocyte-autoantigen interaction requires a secondary nonimmunologic adhesive event.

L11 ANSWER 3 OF 5 MEDLINE  
 ACCESSION NUMBER: 83304098 MEDLINE  
 DOCUMENT NUMBER: 83304098 PubMed ID: 6604367  
 TITLE: [Cellular and humoral immune phenomena in psoriatic arthritis].  
 Untersuchungen uber zellulare und humorale Immunphanomene bei der Arthritis psoriatica.  
 AUTHOR: Neumuller J; Senautka G; Dunky A; Neumann H; Mayer F; Much T; Eberl R; Partsch G  
 SOURCE: WIENER KLINISCHE WOCHENSCHRIFT, (1983 Jun 10) 95 (12) 416-22.  
 Journal code: 21620870R. ISSN: 0043-5325.  
 PUB. COUNTRY: Austria  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: German  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198310  
 ENTRY DATE: Entered STN: 19900319  
 Last Updated on STN: 19900319  
 Entered Medline: 19831021

AB The function of cellular immunity factors (lymphocyte transformation and phagocytosis by polymorphonuclear leucocytes [PMN] and monocytes in connection with the concentration of intracellular ATP) and humoral immunity factors (serum concentration of immunoglobulin and complement factors C'3 and C'4) was investigated in 16 controls, 21 patients with psoriatic arthritis and 19 with psoriasis vulgaris. The results were compared with the clinical and anamnestic data of the patients. PMN phagocytosis of zymosan opsonized with rabbit standard serum was decreased in psoriasis vulgaris in comparison with the controls. Also, monocyte phagocytosis of non-opsonized zymosan was decreased in psoriatic arthritis, as compared with psoriasis vulgaris. Furthermore, in PMNs intracellular ATP was elevated in psoriatic arthritis as compared with the controls, but decreased in comparison with patients with psoriasis vulgaris. The intracellular ATP in monocytes was decreased in psoriasis vulgaris as compared with the controls. Humoral immunological findings: serum IgG concentration was higher in psoriatic arthritis than in controls and in psoriasis vulgaris. Elevated C'3 and decreased C'4 serum concentrations in psoriatic arthritis indicate an activation of the complement system.

L11 ANSWER 4 OF 5 MEDLINE  
 ACCESSION NUMBER: 83153661 MEDLINE  
 DOCUMENT NUMBER: 83153661 PubMed ID: 6830791  
 TITLE: Binding of autologous IgG to human red blood cells before and after ATP-depletion. Selective exposure of binding sites (autoantigens) on spectrin-free vesicles.  
 AUTHOR: Muller H; Lutz H U  
 SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1983 Apr 6) 729 (2) 249-57.  
 Journal code: 0217513. ISSN: 0006-3002.  
 PUB. COUNTRY: Netherlands  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198305  
 ENTRY DATE: Entered STN: 19900318  
 Last Updated on STN: 19900318  
 Entered Medline: 19830527

AB Binding of autologous IgG to fresh, ATP-depleted red blood cells as well as to spectrin-free vesicles was studied by a non-equilibrium binding assay using 125I-iodinated protein A from Staphylococcus aureus. IgG binding was 14-times higher to spectrin-free vesicles than to ATP-maintaining red blood cells and 4-times higher than to ATP-depleted erythrocytes from which these vesicles were released. Protein A binding to vesicles that were released from washed and nutrient-deprived erythrocytes, was dependent on added autologous IgG. However, spectrin-free vesicles that were spontaneously released from erythrocytes conserved in whole blood, bound similar amounts of protein A with or without added autologous IgG (0.45-0.55 ng/micrograms band 3 protein). These findings demonstrate that opsonization of spectrin-free vesicles by autologous IgG occurs not only in the test tube, but also under blood blank conditions. The binding characteristics of IgG to spectrin-free vesicles are indicative of a natural autoantibody rather than an unspecific binding of autologous IgG. The preferential binding of IgG to spectrin-free vesicles implies a selective exposure of corresponding autoantigens in membrane regions that have lost cytoskeletal anchorage and bud off.

L11 ANSWER 5 OF 5 MEDLINE  
 ACCESSION NUMBER: 81000919 MEDLINE  
 DOCUMENT NUMBER: 81000919 PubMed ID: 6157441  
 TITLE: Autoimmune thrombocytopenic purpura.  
 AUTHOR: Karparkin S  
 SOURCE: BLOOD, (1980 Sep) 56 (3) 329-43. Ref: 157  
 Journal code: 7603509. ISSN: 0006-4971.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 198011  
ENTRY DATE: Entered STN: 19900316  
Last Updated on STN: 19970203  
Entered Medline: 19801125

AB Adult autoimmune thrombocytopenic purpura (ATP) is a platelet disorder that develops in certain individuals with a genetic as well as sex (female) predisposition following an environment event (?viral). This results in the production of an IgG antiplatelet antibody capable of reacting with the host's platelets, as well as crossing the placenta. This leads to the rapid clearance and destruction of opsonized platelets by the reticuloendothelial system, particularly the spleen, by greater than tenfold the normal rate. Bound platelet IgG correlates with disease severity, whereas serum antiplatelet IgG does not. It has not been rigorously established whether bound platelet IgG is directed against a platelet antigen or represents an immune complex bound to the platelet Fc receptor. Nevertheless, several lines of evidence suggest that antiplatelet IgG binds directly to a platelet antigen(s). Megakaryocyte number, volume, and mass are increased commensurate with increased platelet turnover. Platelets of increased size, megathrombocytes, are noted on peripheral smear or via platelet volume distribution analysis. Megathrombocyte number is proportionate to megakaryocyte number and to platelet turnover. Megathrombocyte diameter is inversely proportional to platelet survival. Antiplatelet antibody is also associated with qualitative platelet functional defects, which are indistinguishable from those noted with thrombopathia (i.e., apparent platelet release defect). Antibody-induced functional defects are probably more common than quantitative thrombocytopenic defects and may represent a significant portion of those women with the "easy bruising" syndrome and normal platelet count. Adults who develop ATP generally develop the chronic variety, which remains permanently with the patient. Treatment should be directed towards maintaining the patient free of purpura, not restoring the platelet count to normal. This can generally be accomplished with a platelet count of > 40,000/cu mm with patients having this disorder. Approximately 50% of patients respond to steroids by a significant elevation of platelet count and improvement of purpura. However, cessation of therapy results in eventual relapse if the disease is of the chronic variety. Splenectomy is successful in approximately 65-75% of patients, resulting in a restoration of the platelet count to normal or safe levels by removing a major source of platelet destruction as well as antibody production; platelet survival improves. At least 50% of patients "in remission" following steroids or splenectomy generally have a compensated thrombocytolytic state in which increased platelet production keeps up with increased platelet destruction. Antiplatelet IgG can often be found in the serum of these patients. Patients refractory to steroids and/or splenectomy present with a serious therapeutic problem. Immunosuppressive therapy is effective in approximately one-third of refractory patients, but often relapses occur, requiring maintenance therapy with potentially mutagenic drugs...

=> dis his

(FILE 'HOME' ENTERED AT 20:03:16 ON 25 SEP 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002

L1 1375 S SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU  
L2 304 S L1 AND IMMUN?  
L3 11 S L1 AND (HISTOCOMPATIBILITY OR MHC)  
L4 8 DUP REM L3 (3 DUPLICATES REMOVED)  
L5 0 S (ANTIGEN (IN) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND (  
L6 15 S ANTIGEN (P) ATP (P) PRESENT? (P) APC  
L7 5 DUP REM L6 (10 DUPLICATES REMOVED)  
L8 0 S OPSON? (P) GHOST? (P) ATP  
L9 123 S OPSON? (P) ATP  
L10 45 DUP REM L9 (78 DUPLICATES REMOVED)  
L11 5 S L10 AND ANTIGEN

=> s 19 (P) administ?  
L12 0 L9 (P) ADMINIST?

=> s 16 (P) administ?  
L13 1 L6 (P) ADMINIST?

=> dis l13 ibib abs

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:184925 CAPLUS  
DOCUMENT NUMBER: 136:231233  
TITLE: A method for inducing or suppressing immunity  
comprising phagocytic particles and ATP receptors  
INVENTOR(S): Silverstein, Samuel C.; Loike, John D.; Divirgilio,  
Francesco  
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New  
York, USA  
SOURCE: PCT Int. Appl., 74 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020042	A1	20020314	WO 2001-US28171	20010907
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
PRIORITY APPLN. INFO.:			US 2000-658698	A 20000908

AB The invention provides methods of delivering an antigen to class I or class II MHC receptors to induce immunity against the antigen in a subject having a disease. This invention also provides methods of delivering an antigen to a class I or class II MHC receptor to suppress immunity against the antigen in a subject having a disease. The method comprises: (a) filling particles with the

antigen and ATP resulting in Ag/ATP-filled particles; (b) coating the Ag/ATP-filled particles with a ligand for an antigen-presenting cell; (c) incubating the ligand-coated Ag/ATP-filled particles with ligand-binding antigen-presenting cells under conditions permitting the APCs to bind to the particles and APC phagolysosomes to ingest the ligand-coated Ag/ATP-filled particles to facilitate delivery of the ingested antigen to class I or class II MHC and expression on the surface of the APC; (d) incubating the Ag-APCs of step (c) with lymphocytes previously removed from the subject having the disease; and (e) administering the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the antigen. The antigen-presenting cells can also be administered to induce or suppress immunity against the antigen.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT